
Tbx1 controls the morphogenesis of pharyngeal pouch epithelia through mesodermal Wnt11r and Fgf8a.

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Public Summary:

In this publication, Choe et al use the zebrafish to show how the DiGeorge Syndrome gene Tbx1 coordinates development of the face. In particular, they show that Tbx1 is required to produce two important cell-cell communication proteins, Wnt11r and Fgf8a

Scientific Abstract:

The pharyngeal pouches are a segmental series of epithelial structures that organize the embryonic vertebrate face. In mice and zebrafish that carry mutations in homologs of the DiGeorge syndrome gene TBX1, a lack of pouches correlates with severe craniofacial defects, yet how Tbx1 controls pouch development remains unclear. Using mutant and transgenic rescue experiments in zebrafish, we show that Tbx1 functions in the mesoderm to promote the morphogenesis of pouch-forming endoderm through wnt11r and fgf8a expression. Consistently, compound losses of wnt11r and fgf8a phenocopy tbx1 mutant pouch defects, and mesoderm-specific restoration of Wnt11r and Fgf8a rescues tbx1 mutant pouches. Time-lapse imaging further reveals that Fgf8a acts as a Wnt11r-dependent guidance cue for migrating pouch cells. We therefore propose a two-step model in which Tbx1 coordinates the Wnt-dependent epithelial destabilization of pouch-forming cells with their collective migration towards Fgf8a-expressing mesodermal guideposts.

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